LATERAL MEDULLARY SYNDROME

A previously well 67-year-old man presented to the emergency department (ED) with a 4-hour history of vertigo, nausea, and vomiting. He had a 50-pack-year smoking history and was not taking any medications. There was no history of neck trauma or manipulation. While in the ED, his condition deteriorated. He developed slurred speech, numbness and paresthesias of the left hemibody (with preserved facial sensation), and difficulty swallowing liquids.

His blood pressure was 195/90 mm Hg, and his pulse was regular at 66 beats/min. Cardiovascular, respiratory, and abdominal examinations were all unremarkable, and his mental status was normal. Verbal comprehension, naming, and repetition were normal; however, his speech was dysarthric. Cranial nerve examination revealed right-sided ptosis and miosis, consistent with a partial Horner syndrome. The patient's visual fields were normal on confrontational testing, and his extraocular movements were full and without nystagmus. There was symmetrical contraction of the muscles of facial expression. Pinprick sensation was decreased throughout the right face, and his right corneal reflex was absent. His uvula was deviated to the left, and his gag reflex was absent. There was mild pyramidal pattern weakness in his right arm and leg (flexors weaker than extensors in the arm; extensors weaker than flexors in the leg). Deep tendon and superficial reflexes were normal. Sensation to pain and temperature was decreased below the neck on the left side. Coordination assessment with finger-to-nose and heel-to-shin testing revealed severe right-sided dysmetria. When sitting or standing, the patient had marked truncal ataxia, with a tendency to fall to the right. He was unable to ambulate.

Routine laboratory investigations and an electrocardiogram were normal. An unenhanced (noncontrast) computed tomographic (CT) scan of the brain was normal. The diagnosis of lateral medullary infarction was made on clinical grounds. Acute treatment with intravenous tissue plasminogen activator was not offered due to the delayed presentation (clinical assessment was completed 6 hours following symptom onset). Magnetic resonance angiography (MRA) of the head and neck vessels showed occlusion of the proximal right vertebral artery, suspicious for extracranial dissection. The PICA was patent. With supply through collateral flow via the basilar and left vertebral arteries. Magnetic resonance imaging (MRI) of the brain identified an area of restricted diffusion in the right lateral medulla

Within 48 hours, he improved dramatically. His speech returned to normal, his dysmetria resolved, and he was able to sit unassisted. The patient's dysphagia persisted, requiring placement of a gastric tube. A statin was commenced for management of newly diagnosed dyslipidemia. Smoking cessation counselling was provided. Ten days after admission, the patient was eating modified-consistency meals and was transferred to a stroke rehabilitation centre. At 3 months postdischarge, he had recovered almost entirely (Modified Rankin Scale [MRS] 5 14). He no longer required the gastric tube and was ambulating independently. Repeat neurovascular imaging prior to the 6- month follow-up showed recanalization of the vertebral artery.